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Nutrition and lameness in pasture-fed dairy cattle

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ABSTRACT

This review examines the potential for nutritional factors to contribute to the high incidence of foot lameness in New Zealand pasture-fed dairy cattle. Spring and autumn temperate pastures are highly digestible, contain high concentrations of rumen degradable protein and the effectiveness of neutral detergent fibre (NDF) may be too low to support optimum rumen function. Risk of laminitis and lameness increases with chronic ruminal acidosis and there is evidence for sub-optimal rumen pH in New Zealand cows.

Inadequate uptake of trace elements and minerals may compromise hoof health, and there is evidence for lameness associated with the ingestion of high concentrations of anti-nutritional compounds.

The clinical examination of lameness in pasture-fed cows must incorporate an investigation of the nutritional status of the cow in addition to environmental, management, and animal factors.

Keywords: Lameness; dairy cow; pasture; nutrition

INTRODUCTION

Lameness is widespread in the dairy industry of New Zealand, with a reported annual incidence of 10 – 14% of cows in New Zealand herds (Cagienard, 1973; Dewes, 1978). Lameness results from a combination of predisposing factors, including environment, management, conformation of cows and nutrition (Chesterton et al., 1989). Nutritional factors moderate the incidence and severity of lameness by contributing to the occurrence of laminitis (pododermatitis aseptica diffusa), yet the incidence of laminitis in New Zealand cows is unknown.

Lesions that are associated with laminitis have been reported for New Zealand cows (Dewes, 1978; Tranter and Morris, 1991; Vermunt, 1992) and the potential for an association between feeding high quality temperate pastures and laminitis has been proposed (Vermunt, 1992; Tranter and Morris, 1991; Tranter et al., 1991; Macky, 1994; Ossent et al., 1997). The purpose of this review is to consider the potential for a relationship between high quality pastures and risk of lameness, particularly laminitis, in New Zealand dairy cows.

AETIOLOGY OF LAMENESS IN NEW ZEALAND COWS

Investigations of cases of lameness in New Zealand dairy herds have demonstrated the high incidence of lesions involving the claw. Lesions of the hoof were responsible for 67% (Dewes, 1978) and 81% (Tranter and Morris, 1991) of lameness. Remaining cases of lameness were attributed to footrot, strains of tendons and joints, injuries and arthritis.

Much lameness research has therefore focused on understanding the development of lesions of the claw. Many factors have been implicated in the onset of foot lameness (Table 1). Limited studies within New Zealand suggest that some but not all factors are relevant for pasture-fed dairy cows (Dewes 1978, Dewes 1979). A case-control study of factors that influenced foot lameness in Taranaki (Chesterton et al., 1989) concluded that risk factors associated with movement of cows to the milking shed and the milking process accounted for 64% of the variation of prevalence of lameness. Cow characteristics explained a further 9.5% of the variance. Importantly, 26.5% of the variance between high and low incidence herds was unexplained by the model (Chesterton et al., 1989). The nutritional history for the high and low incidence herds was not reported, and dietary factors place cows at greater risk of laminitis and foot lameness (Nocek, 1997; Bickert et al., 1997).

TABLE 1: Non-nutritional factors that may moderate the incidence and prevalence of lesions of the foot

<table>
<thead>
<tr>
<th>Environmental / Management Factors</th>
<th>Animal factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poorly maintained race surface</td>
<td>Individual cow susceptibility</td>
</tr>
<tr>
<td>Concrete flooring and yard surfaces</td>
<td>Conformation</td>
</tr>
<tr>
<td>Cows packed too tightly in holding yard</td>
<td>Breed: Friesians and Holsteins more susceptible</td>
</tr>
<tr>
<td>Insufficient exercise for housed cattle</td>
<td>Social confrontational activity</td>
</tr>
<tr>
<td>Too much exercise and excessive walking for pasture fed animals</td>
<td>Oestrus activity</td>
</tr>
<tr>
<td>Use of farm dog in cow handling</td>
<td>Age/Parity: 2 year old cows generally more susceptible</td>
</tr>
<tr>
<td>Impatient farmer hurrying cows</td>
<td>Stage of lactation: More common first 3 months</td>
</tr>
<tr>
<td>Lack of use of footbath (in some cases only)</td>
<td>Hind limbs &gt; fore limbs</td>
</tr>
<tr>
<td>High moisture/rainfall and standing/walking in slurry</td>
<td>Outer claw &gt; inner claw</td>
</tr>
<tr>
<td>Season: Increased lameness in winter and spring</td>
<td>Hoof colour: White feet more lameness than pigmented</td>
</tr>
<tr>
<td>Irregular trimming and overgrown toes</td>
<td>Systemic illness: More lameness with mastitis, retained foetal membranes, udder oedema</td>
</tr>
</tbody>
</table>

1Nilsson (1963); Dewes (1978); Dewes (1979); Bazeley and Pinsent (1984); Chesterton (1989); Tranter and Morris (1991); Tranter et al. (1991); Bergsten (1994); Ossent et al. (1997)

NUTRITION AND LAMINITIS – THE RELATIONSHIP

Laminitis is believed to occur subsequent to the impairment of circulation in the tissues of the foot as a
consequence of multiple, interrelated aetiologies. Altered perfusion of the vasculature compromises the supply of nutrients to horn producing tissues of the digit (Vermunt, 1992). Descriptions of relationships between nutrition and laminitis have been provided elsewhere (Ossent et al., 1997; Nocek, 1997).

Acute laminitis is associated with the engorgement of digital veins and dilation of arterio-venous anastomoses of the lower limb, such that blood is diverted from the peripheral tissues (Ossent and Lischer, 1998). Vasoactive substances may include histamine, lactic acid, serotonin or endotoxins produced during systemic illness, including ruminal acidosis, mastitis and metritis. Ischaemia and anoxia of the corium become evident clinically 4 to 8 weeks after the systemic insult as haemorrhages of the sole, change of the horn colour from white to light yellow, and a softening of the horn tissue most likely as a result of perturbed keratin metabolism.

Sub-clinical and chronic laminitis also occurs subsequent to disturbed nutrient supply to the claw, but is not associated with systemic symptoms of illness. Claw lesions are associated with the production of inferior quality hoof horn and occur 4 to 8 weeks after predisposing nutritional events. Chronic laminitis is characterised by a changed conformation of the hoof. The dorsal wall surface may appear concave with horizontal (growth arrest) lines extending around the circumference of the wall. The sole may appear flattened with overgrowth of the abaxial wall. Chronic laminitis is presumed to occur as a result of repeated nutritional insults to the claw.

While studies have identified nutritional factors that increase the risk of laminitis, including the feeding of rapidly fermentable carbohydrates (Nocek, 1997), it is unclear whether similar disorders can arise on feeds lower in starch, including the pasture dominant diets of New Zealand dairy cows. International studies have also implicated low concentrations of effective fibre and high concentrations of rumen degradable protein as factors increasing risk of laminitis and the use of buffers, increased intake of copper, zinc, and biotin as factors reducing the onset and severity of laminitis.

NUTRITIONAL CHARACTERISTICS OF NEW ZEALAND PASTURES: A LINK WITH LAMINITIS?

Pasture characteristics and rumen function

Acute and chronic rumen acidosis has been linked with laminitis and a greater risk of lameness (Nocek, 1997). In an international review of 20 studies of pasture fed cows, De Veth and Kolver (2001) reported a mean ruminal pH of 6.16, with a range of 5.6 to 6.7. A ruminal pH of 6.4 to 6.8 is considered desirale for optimum cow health and performance (Erdman, 1988). In the absence of detailed data on the ruminal pH for New Zealand cows, investigation of pasture quality data lends insight as to the potential of sub-optimal ruminal pH. In recent years, there has been an increased amount of information provided on the nutritional composition of New Zealand pastures (Moller, 1996; Kolver, 1998; Kolver, 2000). This information indicates that ryegrass/clover pastures in the winter, autumn and early spring period are high in water content, relatively low in fibre and high in protein compared to the summer period.

(1) Pasture Fibre

An adequate intake of neutral detergent fibre (NDF) and acid detergent fibre (ADF) is necessary for the maintenance of a ruminal pH within the normal range (Jung and Allen, 1995; Mertens, 1997). The NDF and ADF concentrations described for New Zealand pastures (Table 2) indicate that these can be at a concentration consistent with the lowest range of NDF and ADF required for rumen stability. The NRC (1989) recommends a minimum of 25% NDF in the total dietary DM, 75% of which is supplied by coarse forage, to maintain rumen function and health.

TABLE 2: Range of dietary variables for spring and autumn temperate pastures

<table>
<thead>
<tr>
<th>Factor</th>
<th>Range</th>
<th>Optimum for early lactation dairy cows</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dry Matter %</td>
<td>12 - 18</td>
<td>50 – 60</td>
</tr>
<tr>
<td>Crude Protein</td>
<td>14 – 32</td>
<td>18</td>
</tr>
<tr>
<td>Neutral Detergent Fibre</td>
<td>25 – 48</td>
<td>20 (CPM Dairy); 17 (Kolver, 2000); 22 (Mertens, 1997)</td>
</tr>
<tr>
<td>Effective Neutral Detergent Fibre</td>
<td>10 – 24%</td>
<td>21</td>
</tr>
<tr>
<td>Acid Detergent Fibre</td>
<td>18 – 36</td>
<td></td>
</tr>
<tr>
<td>Non-Structural Carbohydrate</td>
<td>8 – 20</td>
<td>36</td>
</tr>
</tbody>
</table>

‘Optimum’ NDF and ADF values for rumen function must be interpreted with care because these do not adequately predict a feed’s ability to maintain a stable rumen pH. The alternative terms ‘effective NDF’ (eNDF) and ‘physical eNDF’ (peNDF) better define the physical characteristics of fibre. Effective NDF from long forages has important physical effects, including the maintenance of normal ruminal pH, the encouragement of chewing and rumination, the formation of the floating rumen mat of large particles on the liquid pool of rumen contents and the stimulation of rumen motility.

Mertens (1997) defined a minimum peNDF intake of 22.3% of the ration DM to maintain a base rumen pH of 6.0, while New Zealand Dairy Research Corporation recommendations are lower, at 17% of DM (Kolver, 2000). An accurate estimate of eNDF and peNDF for New Zealand perennial ryegrass/white clovers remains poorly defined. Kolver (1998) proposed an eNDF for high quality pasture of between 40 and 50% of total NDF. These estimates should be viewed as preliminary. Under some conditions (Table 2) the diet of New Zealand pasture-fed dairy cows may contain less than 20% eNDF and cows will be at greater risk of rumen acidosis.

(2) Pasture Moisture Content

Temperate grasses are frequently characterised by a low content of dry matter (DM) (Table 2) and the ingestion of high moisture feeds is associated with the reduced addition of saliva per kg DM eaten (Meyer et al., 1964). Saliva produced during eating and rumination is an essential mechanism by which ruminal pH is maintained within a normal range through the addition of ruminal buffers,
primarily sodium bicarbonate and disodium phosphate (Erdman, 1988). Mechanisms of reduced flow are however complex and may be associated not only with a high forage concentration of water but also a low NDF or eNDF.

(3) Pasture Protein

The pastures of Australasian dairying systems are characterised by high levels of crude protein (CP), particularly during the spring and autumn months (Table 2). The digestion of CP in high quality lush pastures is characterised by rapid ruminal degradation, substantial losses of N between mouth and duodenum (Beever et al., 1986) and high concentrations of rumen and blood ammonia and urea.

High concentrations of rapidly degradable protein may modify risk of laminitis through one of two mechanisms. High levels of rumen ammonia may ‘buffer’ changes in rumen pH. A rapid association of ammonia with hydrogen ions removes hydrogen from solution and may neutralise up to 10-15% of VFA produced (Owens, 1998).

Conversely, the potential for negative associations between feeding highly degradable protein and the severity of laminitis in cattle has been reported (Bazzely and Pinsent, 1984; Bargai et al., 1992; Ossent et al., 1997). The toxic effects of ammonia are well described (Bruere et al., 1990) therefore high concentrations of blood ammonia and/or urea may compromise the sensitive germinial cells of the lamellae and corium. Alternatively, the ruminal conversion of the amino acid histidine to histamine may increase blood concentrations of histamine or histaminosis may arise as a result of a hypersensitivity to allergenic dietary proteins (Nilsson, 1963). Histamine has been implicated as a contributing factor to perturbed supply of nutrients to horn producing tissues.

An inadequate supply of the sulphur-containing amino acids methionine and cysteine to the corium may increase incidence of lameness as a result of the formation of soft horn. The use of methionine supplements has been advocated to improve the integrity of hoof keratin (Greenough, 1985) however evidence of an association between methionine supplementation and hoof hardness is lacking (Clark and Rakes, 1982). Ryegrass contains a relatively high concentration of methionine compared to many feeds 3.12% (95% confidence interval 2.21-4.03%) of the amino acids in ryegrass (Lean, unpublished).

(4) Nitrogenous Fertiliser and Pasture Nutrition

Nitrogenous fertilisation promotes the growth of a high quality, moist lush pasture (Peyraud et al., 1998) that could accentuate the risk of laminitis through a reduced ruminal pH. Studies have however failed to demonstrate a relationship between increased N and reduced rumen pH (Van Vuuren et al., 1992; Mackle et al., 1996, De Visser et al., 1997). Relationships between use of N fertiliser and rumen function will be determined by time from application to grazing, grazing management and other components of the diet.

INDICATORS FOR SUB-OPTIMAL RUMEN FUNCTION ON HIGH QUALITY PASTURES

(1) Feed Characteristics

An objective and subjective investigation of the chemical and physical characteristics of a herd’s diet may provide evidence for sub-optimal rumen function. Assessment of DM %, digestibility, energy, non-structural carbohydrate content, NDF and ADF will provide an overview of feed quality while visual appraisal will give a subjective assessment of physical attributes that may influence rumen function. Risk of ruminal acidosis is greater when pasture is lush, leafy and fast growing, and following N application. A high leaf to stem ratio and low shear strength may be considered indicative for an increased risk of acidosis, particularly for autumn/spring regrowth pastures.

The low pH of silages (pH < 4.0), particularly maize and whole crop cereal silages may increase risk of ruminal acidosis, particularly when fed in conjunction with lush high quality pastures. It is important to recognise that pre-formed lactic acid in silage can contribute to a lower ruminal pH. The pathogenesis of acidosis may be influenced by a combination of de novo synthesis of acids in the rumen from precursors and by an acid ‘dump’ associated with silage feeding. This is particularly relevant when considering maize silage. Chop length of forages will further mediate ruminal pH, because short chop silage and hay reduce chewing and rumination time and therefore saliva production. Starchy cereal grains, particularly wheat, can be rapidly rumen fermentable, tend to encourage a lactic acid fermentation and may increase risk of acidosis (Opatpatanakit et al., 1994).

(2) Animal Factors

RUMEN pH

The quantification of ruminal pH from a sub-sample of a herd will assist with the diagnosis of ruminal acidosis. However ruminal pH may have returned to normal when laminitic lesions are first diagnosed as a result of the 4 to 8 week delay between nutritional insult and the onset of laminitis.

Several methods have been reported for the collection of ruminal fluid.

Ruminal cannula

Collection of rumen contents from cattle with an indwelling ruminal cannula remains the preferred method for the determination of rumen pH however the impractical nature of rumen cannulae limits their use in commercial dairy situations.

Rumenocentesis

The collection of rumen fluid using percutaneous needle aspiration is a more practical method for the determination of ruminal pH. A full description of the rumenocentesis method was provided by Garrett et al. (1999). This method, however, may not be benign as rumenocentesis of healthy cattle in a trial examining acidosis found milk yields that were measured twice daily fell markedly for 24-48 hours after sampling (Lean, unpublished).

Stomach tubing

Samples of ruminal fluid taken by stomach tube have a higher pH than those obtained through a rumen cannula.
(Erdman, 1988), most likely as a result of salivary contamination, therefore ruminal pH samples collected by stomach tube should be interpreted with caution.

**Numbers of cows to sample**

Samples of rumen fluid should be collected from a minimum of 6 – 12 cows to provide an overview of herd status (Olson, 1997; Garrett et al., 1999). Where concentrates or grains are being fed separately from forages, samples should be taken 2 to 4 hours following feeding of the concentrate. For grass only or total mixed ration herds, collect samples 4 to 8 hours after feeding.

There is a high likelihood of ruminal acidosis in a herd when more than 30% of sampled cows have ruminal pH of 5.5 or less (Olson, 1997). A ruminal pH of 5.6 to 5.8 is suggestive of a marginal or developing problem of ruminal acidosis, while a pH of > 5.9 was considered ‘normal’ (Olson, 1997). There is a requirement to validate these ranges under New Zealand conditions.

**MILK FAT CONCENTRATION**

Repeated assessment of bulk vat concentration of milk fat % may under some situations provide an indirect predictor for ruminal acidosis because ruminal pH is positively associated with milk fat concentration. The relationship between ruminal pH and milk fat concentration is not absolute under all conditions, being confounded by stage of lactation, fat content of diet and body fat mobilisation. A reduction in concentration of milk fat should be considered indicative only for low ruminal pH, and must be considered in conjunction with other objective assessments of rumen function.

**CHEWING TIME**

Chewing time is positively associated with ruminal motility (Norgaard, 1989) and pH (Armentano and Pereira, 1997). Chewing time has therefore been proposed as a potential indicator of the physical effectiveness of fibre. About 50% of cows lying down after feeding should be chewing, a lesser proportion may be indicative of disturbed rumen function (Troutt, 1991). The relationship between chewing time and effectiveness of fibre can however be confounded by animal factors, including liveweight and feed intake level, and does not adequately account for intrinsic acidotic properties or buffering capacity of a feed. The use of chewing activity as a predictor for perturbed rumen function is a useful, non-invasive, ‘on farm’ indicator, but must be considered together with other animal and feed measures.

**FAECAL CHARACTERISTICS**

The faeces of cows can provide indirect evidence of clinical and sub-clinical acidosis. Faecal moisture content is increased as a result of increased osmolality of acidic rumen and gastrointestinal contents and faeces may contain increased proportions of undigested feeds as a result of reduced cellulolytic microbial activity at a lower ruminal pH. Faecal pH is poorly correlated with rumen pH due to fermentation and buffering in the hindgut (Clayton et al., 1999) therefore faecal pH values should be interpreted with caution.

**MAINTAINING A STABLE RUMEN FERMENTATION**

Management changes that remedy sub-optimal rumen function may reduce the negative effects of diet on incidence and severity of lameness in pasture fed cows.

**ADEQUATE FIBRE**

Where the assessment of feeds and the herd suggests sub-optimal ruminal pH, the provision of a high eNDF supplementary feed source may benefit cow health and productivity. High eNDF feeds (eNDF > 90% of total NDF) include; cereal and ryegrass straw, poor quality pasture silage and hay and summer dry pasture (Kolver, 2000). The supplementation of cows at rates of 1 – 2 kg DM of high eNDF feed / cow / day (Macky, 1994) may improve ruminal function when ruminal acidosis is present or suspected, or where pasture NDF concentration is less than 35 – 40% (Kolver, 2000).

**RUMEN BUFFERS**

Buffers may assist in the maintenance of ruminal pH, particularly where the diet contains a high proportion of cereal silage or cereal grains. Sodium bicarbonate neutralises VFA in the rumen and alters the pH of blood, while magnesium oxide acts as a neutralising agent in the rumen (Erdman, 1988; Kellaway and Porta, 1993). Due to the different modes of actions when used in combination, sodium bicarbonate and magnesium oxide may give some synergistic activity in the rumen. Sodium bentonite is a clay product that may reduce the effects of lactic acidosis, however results from studies show inconsistent outcomes (Kellaway and Porta, 1993). Limestone (calcium carbonate) has no buffering activity in the rumen but may act to regulate pH in the intestines (Hutjens, 1991; Kellaway and Porta, 1993).

The use of buffers should not replace the requirement for high eNDF supplements because sodium bicarbonate has a limited capacity for the control of rumen pH in pasture based diets (Lean et al., 1998, Clayton et al., 1999).

**RUMEN MODIFIERS**

Rumen modifiers including monensin, lasalocid, tylan and virginiamycin have been used successfully to control risk of acidosis (Lean et al., 1998, Clayton et al., 1999). The use of these rumen modifiers also provides useful tools for investigating the pathogenesis of acidosis in cattle fed primarily on pasture. Currently sodium monensin (Rumensin®) is the only rumen modifier currently registered for use in New Zealand adult dairy cattle.

**MACRO AND TRACE MINERALS, VITAMINS AND LAMENESS**

**Copper Deficiency**

A dietary deficiency of copper, either primary (due to an absolute lack of dietary copper) or secondary (resulting from normal or low dietary copper levels interacting with high intakes of sulphates, molybdenum, iron, and zinc) may contribute to a greater lameness primarily through effects on skeletal integrity (Smart, 1985; Smart and Cymbaluk, 1997). Lameness is associated with microfractures of the primary trabeculae of the distal metacarpal and metatarsal
bones. Further, copper deficiency is associated with poor quality horn (Smart and Cymbaluk, 1997) and an increased incidence of infectious pododermatitis (Amstutz, 1985).

**Zinc Deficiency**

Zinc is required for RNA and DNA synthesis, protein metabolism, anti-inflammatory processes and wound healing. Zinc deficiency may occur as a primary dietary deficiency or secondary to excessive intakes of copper, calcium or iron (Graham, 1991) or as a result of depletion following metabolic stress, disease and inflammatory reactions.

A sub-optimal zinc status has been implicated in the excess growth of soft horn and the formation of abnormal hooves, an increased incidence of footrot and interdigital dermatitis and parakeratosis of the lower limb (Smart, 1985; Smart and Cymbaluk, 1997).

**Calcium and Phosphorus Imbalance / Deficiency**

Calcium and phosphorus are interrelated in the mineralisation of osteoid therefore deficiencies are characterised by osteomalacia and pathological fractures. Lameness is typically associated with a gross imbalance of the dietary proportions of calcium and phosphorus, or as an absolute lack of either in the diet.

**Vitamin A or D Deficiency**

The vitamin A status of cattle is not directly linked to hoof lameness however a deficiency of vitamin A may compromise the growth and maturation of cartilage (Smart and Cymbaluk, 1997). Vitamin A deficiency is unlikely to occur in pasture fed cattle due to the abundance of carotene (Frye et al., 1991).

An increased incidence of lameness subsequent to vitamin D deficiency may occur in housed calves, however ultraviolet activation of provitamin A in the skin is typically sufficient for New Zealand young stock housed outdoors.

**Biotin Deficiency**

Biotin is a water-soluble B complex vitamin considered essential for the growth and maintenance of epidermal tissues, including keratin of the claw. Where cattle are fed high energy processed feeds, dietary biotin and the synthesis of biotin by gastrointestinal microflora may be inadequate for optimal health and production. Biotin deficiency is associated with cracked and brittle hoof horn (Vermunt and Greenough, 1995), increasing the incidence of lameness. Supplementation with biotin at 20 mg biotin/cow/day may improve hoof hardness and resistance to wear, however a prolonged period of biotin supplementation (more than 3 to 4 months) is required before beneficial effects are seen (Fitzgerald et al., 2000).

**Selenium Toxicity**

Acute or chronic ingestion of high concentrations of selenium has been associated with horizontal bands and cracks of the hoof and in severe cases, sloughing of the hoof from the corium (Bruere et al., 1990). An accumulation of selenite ions can lead to the replacement of sulphur in essential proteins (Cooper, 1987), possibly contributing to reduced integrity of hoof structure.

Selenosis can result from the ingestion of feeds growing on seleniferous soils, particularly selenium accumulating plants (Bruere et al., 1990). More commonly, selenium toxicity results from the inappropriate prophylactic use of selenium products to prevent selenium deficiency diseases.

**Fluoride Toxicity**

Lameness has been reported for cattle with chronic exposure to fluoride, described as 'moving lameness' most likely due to minor or major skeletal fractures of bones of the hoof (Bruere et al., 1990). High intakes of fluoride have been associated with pastures contaminated by industrial wastes from smelters, volcanic eruptions or pastures recently fertilised with rock phosphate or superphosphate, and water sources containing naturally high levels of fluoride.

**ANTI-NUTRITIONAL FACTORS AND LAMENESS**

**Nitrate Toxicity**

Nitrate toxicity is commonly reported for dairy cows grazing rapidly grown greenfeed crops and new pastures following dry or drought conditions (Bruere et al., 1990). While most cases of nitrate toxicity are acute and characterised by ataxia, dyspnoea and death, there exists a potential relationship between nitrate toxicity and laminitis (Vermunt, 1992). Nitrate is converted to nitrite in the rumen and nitrite converts haemoglobin to methaemoglobin, reducing the oxygen carrying ability of the blood. Nitrite is also a potent vasodilator. Stagnation and pooling of blood in the peripheral circulation, including the vascular beds of the corium may induce anaoxia and the accumulation of tissue toxins, potentially increasing susceptibility to laminitic lesions.

**Mycotoxins**

The production of mycotoxins from fungal damaged conserved feeds or pastures has been implicated as a cause of laminitis (Nilsson, 1963; MacLean, 1965; Vermunt, 1992).

**Ergotism**

Ergot poisoning results from ingestion of alkaloids produced by the fungus *Claviceps purpurea*, which can infect ryegrass, wheat and barley. Infection of the ovary of the plant results in a mass of hyphae (sclerotium) in the mature seed. Ingestion of the infected seed by cattle can result in lameness, swelling of the hind limbs and loss of extremities (Burfening, 1973) as a result of vasoconstrictor actions of at least three ergot alkaloids (Bruere et al., 1990). Ergotism has been reported in New Zealand but is avoided by appropriate grazing management that avoids accumulation of seed heads.

**Fescue Foot**

Lameness in cattle grazing high endophyte tall fescue (*Festuca arundinacea*) is associated with alkaloids produced by the endophyte *Neotyphodium coenophialum*, particularly the ergopeptide alkaloid, ergovaline. Ergot alkaloids are vasoconstrictive compounds that compromise blood flow to the extremities of cattle, including the hoof (Bruere et al, 1990). Swelling and hyperaemia of the coronary band precedes the development of a dry gangrene of the hooves. Sequelae include hoof deformities and sloughing of the hoof.
Tall fescue grasses currently marketed in New Zealand are endophyte free and are not associated with fescue foot however wild type high endophyte tall fescue remains in limited areas of Northland, New Zealand. New tall fescue endophytes that do not produce the ergot alkaloids are currently being developed and will be available within the next 5 years.

Perennial Ryegrass Endophyte

Alkaloids produced by the perennial ryegrass endophyte (Neotyphodium loli) have been associated with health problems in grazing animals including perennial ryegrass staggers (Bruere et al., 1990) and heat stress (Fletcher, 1998). Ergovaline is the most potent and abundant of the ergopeptide alkaloids through its effects as a dopamine agonist and a vasoconstrictor. Anecdotal evidence exists for horizontal ‘hardship’ lines on hooves coincident with late summer/autumn intakes of high endophyte ryegrass and Fletcher (1998) reported unexplained lameness and swelling around the fetlock in cattle grazing high endophyte ryegrass. Production of ergovaline from different ryegrass cultivar/endophyte associations is variable, and some cultivars produce less ergovaline than others. New perennial ryegrass endophytes that do not produce ergovaline (‘AR1’) are in now available in New Zealand.

CONCLUSIONS

Lameness of dairy cattle results from the effects of many factors, including cow conformation, management, environment, climate and nutrition. Under New Zealand conditions, the majority of cases of claw lameness are attributable to non-nutritional causes (Chesterton et al., 1989). Evidence exists however for sub-optimal rumen function, poor trace element status and the presence of anti-nutritional factors that may moderate the incidence and severity of lameness for pasture-fed cows. There is sufficient evidence to suggest the need to investigate rumen function for pasture-fed New Zealand cows and to determine the potential for a relationship between acute and chronic ruminal acidosis, sub-clinical laminitis and lameness in cattle fed primarily on pasture.

REFERENCES

Bruere, A.N.; Cooper, B.S.; Dillon, E.A. 1990: In: Veterinary Clinical Toxicology. Foundation for continuing education of the New Zealand Veterinary Association Publication Number 127
Chesterston, R.N.; Pflifer, D.U.; Morris, R.S.; Tanner, C.M. 1989: Environmental and behavioural factors affecting the prevalence of foot lameness in New Zealand dairy herds – a case control study. New Zealand veterinary journal 37: 135-142
CPM Dairy Version I.0. Cornell-Penn-Miner. The Centre for Animal Health and Productivity, School of Veterinary Medicine, University of Pennsylvania; The Department of Animal Science, Cornell University; and the William H. Miner Agricultural Research Institute.
Dewes, H.F. 1979: Transit-related lameness in a group of dairy heifers. New Zealand veterinary journal 27: 45
Fletcher, L.R. Endophyte on the dairy farm, is it a problem? Proceedings of the 15th annual seminar of the Society of Dairy Cattle Veterinarians of the New Zealand Veterinary, Association pp. 119-132


MacLean, C.W. 1965: Observations on acute laminitis of cattle in South Hampshire. *Veterinary record* 77: 662-672

Mertens, D.R. 1997: Creating a system for meeting the fibre requirements of dairy cows. *Journal of dairy science* 80: 1463-1481


